served in clinical trials of interferon alfa (but not interferon gamma).[18,19] Although acute hypotension is the most common cardiovascular side effect of tumor necrosis factor-$\alpha$,[20] a case of possible cardiomyopathy associated with prolonged treatment with tumor necrosis factor has been reported.[21] The effect of other cytokines that may be secondarily released following IL-2 administration—such as IL-1, IL-6 granulocyte-macrophage colony-stimulating factor, and IL-3—on the heart has not yet been adequately evaluated.[22,23]

We suggest that patients in whom congestive heart failure or a prolonged dependence on vasopressors develops after IL-2 therapy should be evaluated for myocardial damage due to ischemia, myocarditis, or cardiomyopathy. Assessing the levels of creatine kinase and its isoenzymes is helpful in detecting myocardial injury, as serum levels of this enzyme are usually decreased during IL-2 administration (W.E.S., J.H.W., E.R. Ashwood, MD, unpublished data, January 1989). Rises in the MB fraction may be observed following cardiac injury in IL-2–treated patients.[4,11] Electrocardiograms should be done to exclude ischemic changes. Echocardiograms may be useful in distinguishing between segmental wall motion abnormalities more typical of ischemia and global defects that represent diffuse myocardial processes (myocarditis and cardiomyopathy). Finally, because sepsis can present with hypotension, a search for infection should always be undertaken. We recommend the consideration of right-sided heart catheterization with myocardial biopsy in any patient receiving interleukin-2 in whom unexplained, persistent myocardial dysfunction develops. With this approach, the correct diagnosis can frequently be established and the possibility of infarction or ischemia can be reasonably excluded.

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# Idiopathic Mediastinitis With Superior Vena Cava Obstruction, Cardiac Tamponade, and Cutaneous Vasculitis


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WE DESCRIBE the case of a young woman who presented with cardiac tamponade and in whom a superior vena cava syndrome later developed, associated with a nodular, ulcerative cutaneous vasculitis. This case emphasizes the spectrum of fibrosing mediastinitis. Three similar cases of idiopathic fibrosing mediastinitis with vasculitis have been previously reported.[1-3] Systemic and localized vasculitis have been recognized more commonly in cases of retroperitoneal fibrosis and should be similarly recognized as part of the spectrum of fibrosing mediastinitis.[2,4]

## Report of a Case

A 33-year-old woman was seen in October 1987 because of malaise and abdominal pain for a month, followed by cough, exertional dyspnea, and fever. The patient smoked cigarettes but otherwise was previously in good health and had taken no medications. On initial examination she was febrile and appeared ill with distended neck and superficial chest veins, supraclavicular adenopathy, a pericardial rub, hepatomegaly with a positive Kussmaul's sign, and a 20-mm pulsus paradoxus.

Leukocytosis, anemia, thrombocytosis, and a greatly increased sedimentation rate were noted. A chest x-ray film and echocardiogram revealed a large pericardial effusion. Signs of cardiac compression progressed; pericardiocentesis relieved the tamponade and yielded 300 ml of a sterile exudate with a low leukocyte count.

A computed tomographic (CT) scan of the chest (Figure 1) showed a diffuse nodular mediastinal and left hilar infiltrate suggestive of lymphadenopathy. An abdominal CT scan

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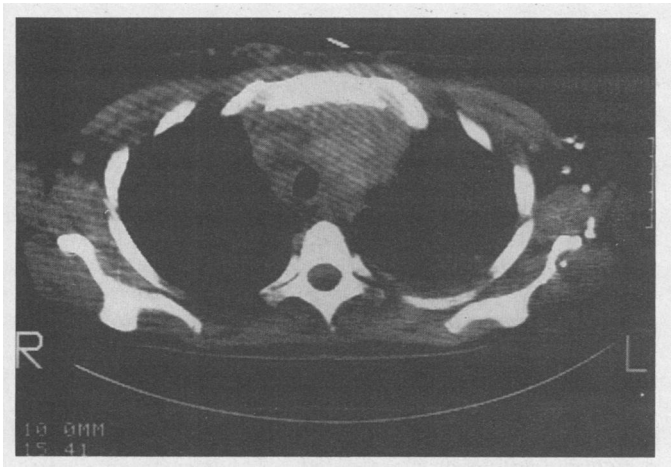


**Figure 1.**—A chest computed tomographic scan in October 1987 shows prominent enlargement of the upper anterior mediastinum with diffuse replacement of fat by soft tissue density infiltrate. Also seen are dilated left chest wall collaterals.

revealed hepatomegaly and pronounced periaortic and mesenteric adenopathy. A bone marrow aspirate revealed granulocytic hyperplasia. On mediastinoscopy the anterior mediastinal space was obliterated by dense adhesive inflammatory tissue. There was nonspecific reactive change of the supraclavicular and paratracheal nodes. Through a limited thoracotomy, a pericardial window was created, revealing an organizing fibrinous pericarditis. A biopsy specimen of the left lower lung showed a nonspecific chronic interstitial pneumonia and marked organizing pleural fibrosis.

The patient was anergic to a battery of antigens. Cultures of blood, marrow, lung, and lymph nodes were negative for bacteria, fungi, and acid-fast bacilli. Autoantibodies were not found; complement components were normal. Serologic tests for histoplasmosis, human immunodeficiency virus, *Coccidioides immitis* and *Legionella* and *Mycoplasma* species were negative. Epstein-Barr and cytomegalovirus serologic tests indicated past infection.

A regimen of methylprednisolone sodium succinate, 80 mg per day, was instituted parenterally, and systemic signs rapidly resolved. Prednisone therapy, 1 mg per kg per day, was continued as an outpatient. As the prednisone dosage was tapered to 20 mg per day, however, edema of the extremities, fatigue, and distension of the upper thoracic superficial veins increased; livedo reticularis and tender, erythematous nodules of the right hand, right buttocks, left thigh, and left ankle developed; and the lower extremity nodules ulcerated.

In June 1988, a magnetic resonance image of the chest showed superior vena caval obstruction with reconstitution caudally through the azygos vein and a large amount of adenopathy of superior mediastinal, precarinal aortopulmonary nodes. The pericardial effusion and hilar and left lower lobe infiltrate had resolved. An abdominal CT scan was now normal. Skin biopsies of the left foot and thigh revealed acute and chronic vasculitis involving arterioles (Figure 2).

Despite the development of ulcerative cutaneous nodules and symptoms of superior vena caval obstruction, the patient had no recurrence of serositis or fever. Hemogram and chemistry panel values and renal function were normal. The sedimentation rate was again increased. The prednisone dosage was increased to 60 mg per day, and azathioprine, 100 mg per day, was added. The lower extremity edema rapidly cleared. Cutaneous nodules regressed slowly and healed. The prednisone dosage was tapered slowly. Signs of the superior vena cava syndrome persisted despite several months of therapy with prednisone and azathioprine.

A CT of the chest in August 1988 revealed clearing of the originally noted mediastinal infiltrate compared with the appearance in October 1987, but numerous collateral veins of the chest, mild to moderate infiltration of the mediastinal fat with stranded and patchy densities consistent with fibrotic change, and obstruction of the superior vena cava were observed. It was elected to institute penicillamine therapy and to discontinue azathioprine. The penicillamine dosage was gradually increased to 750 mg per day while the prednisone dosage was maintained at an average of 20 mg per day. A chest CT scan in May 1989 revealed similar mediastinal changes compared with the August 1988 chest CT scan (Figure 3).

Penicillamine therapy was continued from October 1988 to September 1989. As the prednisone dosage was reduced to 10 mg per day, the cutaneous nodules progressed. Symptoms of the superior vena cava syndrome continued. In September 1989, penicillamine was discontinued and a regimen of hydroxychloroquine sulfate, 400 mg per day, was insti-

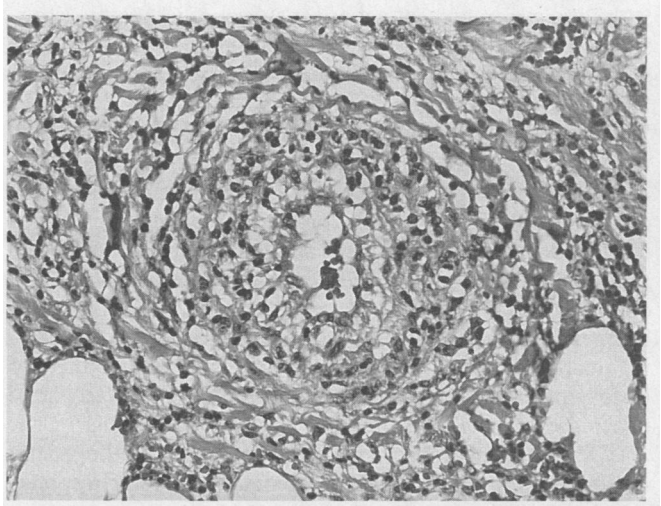

**Figure 2.**—An arteriole in the subcutaneous portion of a skin biopsy shows vasculitis with transmural infiltrate of inflammatory cells, predominantly lymphocytes and histiocytes (hematoxylin and eosin, original magnification × 250).

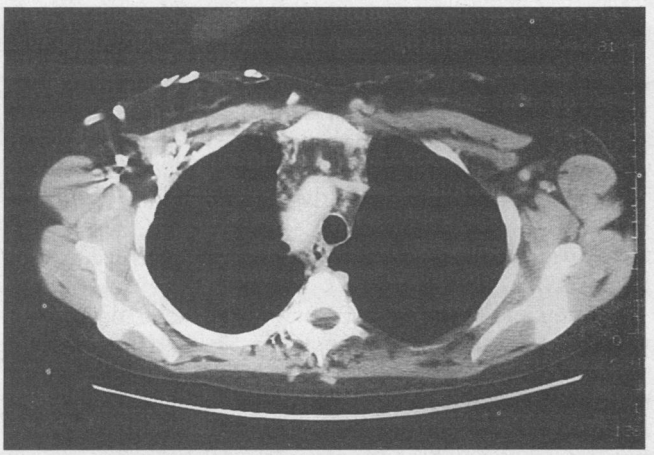

**Figure 3.**—A chest computed tomographic (CT) scan April 1989 shows compression of the superior vena cava and streaky fibrofatty mediastinal densities suggestive of fibrosis. Clearing of the mediastinum compared with the previous chest CT scan and chest wall collaterals are also evident.

tuted. Hydroxychloroquine therapy was discontinued after ten months because of a lack of effect on the cutaneous vasculitis.

In September 1990, the patient presented with recent gastrointestinal bleeding and severe anemia. An upper gastrointestinal endoscopy revealed varices of the proximal esophagus ("downhill varices"). Venography of the upper extremities showed total occlusion of both subclavian arteries as they drained into the superior vena cava, with abundant collaterals around the sites of obstruction. In addition, lower extremity venography and abdominal ultrasonography showed that the inferior vena cava was obstructed from the level of the confluence of the iliac arteries to just below the lower margin of the liver. The ureters were not obstructed.

As of August 1991, the patient is being treated with a regimen of hydrochlorothiazide and prednisone (10 to 20 mg a day) with methotrexate (7.5 mg a week) to control cutaneous vasculitis. There have been no other signs of systemic progression of her disease.

## Discussion

We describe a case of a young woman with an acute, systemic inflammatory process who presented with an effusive, constrictive pericarditis that responded to pericardial stripping and corticosteriod therapy. Within the first six months of this disease process, she had the development of a superior vena cava syndrome due to extrinsic compression from mediastinitis, livedo reticularis, and an ulcerative, nodular cutaneous vasculitis. The patient has been observed for nearly four years and remains stable with a mild, compensated superior vena cava syndrome and limited cutaneous disease.

This patient clearly had an obliterative inflammatory process involving the mediastinum that caused obstruction of the superior vena cava. No drug, infectious, or neoplastic cause was found. As such, this process seems to fit best the spectrum of diseases known as "fibrosing mediastinitis," "sclerosing mediastinitis," or "idiopathic mediastinal fibrosis" in which any structure in the mediastinum, including trachea, esophagus, pulmonary artery, pulmonary vein, and superior vena cava, may become obstructed.[5] The frequency of the superior vena cava syndrome in fibrosing mediastinitis has in fact been found to be from 2%[6] to 12%[7] to 23%[8-10] of reported series.

The occurrence of a vasculitis, as seen in this case, in association with fibrosing mediastinitis has not been commonly recognized or emphasized. In a recent review of pulmonary vascular obstruction with fibrosing mediastinitis, the authors did not include systemic vasculitis.[11] In our review of the English language literature, we have found three previously reported cases of vasculitis in association with fibrosing mediastinitis.[1-3] As in our case, in each of these cases, ulcerative cutaneous nodules were described as features of the vasculitis.

Carton and Wong in 1969 first noted the association of vasculitis with fibrosing mediastinitis.[1] In this case, mediastinal and retroperitoneal fibrosis occurred in a 29-year-old man with superior vena caval obstruction. Arteritis was found in the retroperitoneum, sympathetic vessels, and peripheral small arteries and arterioles of the skin.

In 1981 Littlejohn and Keystone described the case of a young man with mediastinal and retroperitoneal fibrosis and a systemic vasculitis histologically found to involve the skin and mediastinum.[2] Pleuritis, pericarditis, and arthritis were also noted in this patient. In 1986 Fischer and DeLauney similarly described the case of a 29-year-old man with superior vena cava syndrome, livedo reticularis, and ulcerative cutaneous vasculitis. This patient also had retroperitoneal fibrosis.[3]

In our patient, the presenting problem was pericardial tamponade. This is the first report of cardiac tamponade as the initial manifestation of idiopathic fibrosing mediastinitis. In fact, Barrett has reported that mediastinal fibrosis does not invade the pericardium.[12] Others, however, have noted the presence of pleural and pericardial effusions in patients with fibrosing mediastinitis, including the case described by Littlejohn and Keystone and three other cases of mediastinitis without vasculitis in which pericardial thickening or pericardial effusions were noted.[2,13-15] Pericardial disease clearly should be recognized in the spectrum of fibrosing mediastinitis.

The concomitant occurrence of mediastinitis, serositis, and vasculitis can probably be better appreciated by considering the relationship of idiopathic fibrosing mediastinitis to idiopathic retroperitoneal fibrosis. Koep and Zuidema reviewed nearly 500 cases of retroperitoneal fibrosis and found a 3.3% incidence of mediastinal fibrosis.[4] In fact, all three of the cases of mediastinitis with vasculitis noted earlier were also associated with retroperitoneal fibrosis. The recognition that inflammatory fibrosis may occur in more than one organ system or region has led some authors to consider these to be a family of disorders known as "multifocal fibrosclerosis" or "systemic fibrosclerosing syndromes."[1,3,15] Ureteral obstruction from the retroperitoneal process has not developed in our patient, but she was later found to have an inferior vena caval obstruction. She did initially present with periaortic and mesenteric adenopathy and clearly also has had a retroperitoneal inflammatory process.

As to the relationship of vasculitis to retroperitoneal fibrosis, Littlejohn and Keystone noted a 7% prevalence (37 cases) of histologically defined vasculitis in a literature survey of about 500 cases of retroperitoneal fibrosis.[2,4] Of these 37 patients, 30 (81%) had vasculitis in both retroperitoneal and distal sites. The authors suggested that the combination of retroperitoneal and mediastinal fibrosis with cutaneous vasculitis may represent a distinct subset of idiopathic fibrosis seen in young men. The case reported herein of a young woman with mediastinitis and cutaneous vasculitis seems to fall within the spectrum of this subset.

Autoimmune diseases and several other idiopathic, immune-mediated inflammatory disorders have also been associated with retroperitoneal fibrosis. The list of associated disorders includes systemic lupus erythematosus, scleroderma, eosinophilic fasciitis, amyloidosis, and crescentic glomerulonephritis.[16-23] These interrelationships point to a common immune-mediated pathogenesis. Fibrosing syndromes have been linked to other causes including underlying malignancy, infection (particularly granulomatous), a history of drug ingestion (methysergide or other vasoactive pharmacologic agents), trauma, and atherosclerosis.[15,17]

Clearly many causes may trigger similar pathogenetic mechanisms resulting in an inflammatory cellular response and fibrosis. In situations, however, such as our case in which no antigen or defined triggering event can be identified, the precise mechanism of fibrosis and inflammation can only be speculative. Nevertheless, this case serves to empha-

size the breadth of associations of mediastinitis, in particular to add to the recognition of the link between vasculitis, cardiac tamponade, and superior vena caval obstruction.


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# Home Treatment of Calf Deep Venous Thrombosis


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TRADITIONAL THERAPY in the United States for deep venous thrombosis confined to the calf involves seven to ten days of stay in hospital, but British practice advocates only three days.[1] Recent Canadian experience advocates five days of hospital stay to begin anticoagulant treatment.[2] An otherwise healthy 37-year-old man was successfully treated at home for seven days with a continuous intravenous infusion of heparin. There were no complications during or following treatment, and the patient recovered uneventfully. Cost savings of approximately $4,000 were realized by avoiding admission to hospital.

(Callister CB, Wood SD, White GL Jr, Tucker ED: Home treatment of calf deep venous thrombosis. West J Med 1991 Sep; 155:299-300)

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## Report of a Case

The patient, a 37-year-old healthy, nonsmoking, male graduate student, was referred to our family practice clinic for the evaluation of left lower leg pain of a week's duration. His history did not reveal trauma, overexertion, periods of prolonged immobility, or a surgical procedure. He had, however, been admitted to hospital 15 years ago for bilateral thrombophlebitis of the upper legs. He suffered no sequelae from this episode and had had no medical problems until now.

On physical examination, his left calf was exquisitely tender with a positive Homan's sign. Superficial varicose veins were noted on the posterior aspects of both legs. The left calf was without erythema or swelling and did not appear excessively warm to touch. Calf diameter measurements were equal. Femoral, dorsalis pedis, and posterior tibialis pulses were strong and equal bilaterally. The lungs were clear to auscultation, and the heart was normal.

A Doppler ultrasonogram of the left leg revealed the paired peroneal veins in the left calf to be thrombosed. The thrombosis was confined to the peroneal veins. A diagnosis of deep venous thrombosis of the left calf was made. The patient was determined to be a good candidate for outpatient therapy and was willing to try it (Table 1). A home infusion therapy company (New England Critical Care, Westboro, Massachusetts) was contacted to arrange treatment. The initial home nursing visit consisted of a physical assessment; baseline prothrombin time, partial thromboplastin time, and complete blood count; and patient education. Potential problems and side effects related to deep venous thrombosis and anticoagulation therapy were discussed with the patient. After obtaining signed, informed consent, we established venous access in a peripheral vein, and a portable controlled infusion device (Pharmacia Deltec CADD-1) was attached for anticoagulation therapy. Heparin therapy was initiated with a 5,000-unit bolus, followed by 1,000 units per hour by continuous intravenous infusion. Oral anticoagulant medication—warfarin sodium, 10 mg once a day—was also started simultaneously. The nurse made twice-a-day visits to the patient's home to monitor progress and obtain blood specimens.

The laboratory results were reported to the physician, and appropriate changes in heparin dosage were made to keep the partial thromboplastin time between 1.5 and 2.0 times the control value. Similarly, the warfarin dosage was adjusted to keep the prothrombin time in the accepted therapeutic range of 1.2 to 1.5 times the control value.

The patient completed seven days of therapy in this manner. Clear lines of communication between the home infusion therapy team and the attending physician were maintained. The patient's venous access remained patent and without inflammation throughout treatment. He had no adverse effects from heparin and fully recovered from his calf vein thrombosis. Maintenance warfarin therapy was continued for three months with periodic monitoring at the clinic to